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presented by the author

—BY—

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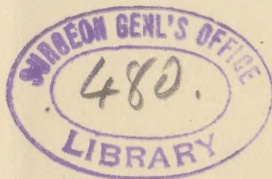


THE TRUE PATHOLOGY OF SUD-
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The committee on business of last year, in formulating this subject for discussion, have undoubtedly sought to limit it to those cases of sudden death in acute pneumonia which seem to be disconnected from any pre-existing or merely accidentally intercurrent fatal morbid process, or complication. A patient passing through acute pneumonia, with previously existing chronic degenerative nephritis may suddenly be seized with uræmic convulsions, coma, and death; or a sufferer from chronic rheumatic endarteritis of the cerebral vessels may during a fit of coughing in pneu-

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monia be attacked by a sudden cerebral hæmorrhage and die. Such cases do not enter the present inquiry. Their pathology is beyond controversy and there is no preventive means, which may reach them, within our present knowledge.

The class of cases under discussion can best be exemplified by the short recital of one typical case, that of our lamented friend and colleague, the late Dr. Woodhull, of Newark, for a history of which I am indebted to the kindness of Drs. Edgar Holden and Charles Young.

This is Dr. Holden's statement:—"Dr. Addison W. Woodhull, born August, 1831. Died in the Spring of 1876, age 45. One morning he said to me on the street: 'I have a very annoying pain in my left side to-day; I wonder if my old wound is going to trouble me.' He went to New York and before his return took a Turkish bath, and on his return home felt miserable but went on with work; was taken at night with a chill and slight dyspnœa; this was followed by fever, cough and soon rusty colored sputa. Dr. O'Gorman and myself

attended him; rapid consolidation of the lung (I think the left) followed.

“The disease ran the usual course of a unilateral pneumonia in a man with sound heart, for I examined not only then, but I before had examined him for life insurance, and at that time found no impairment of any organs, save the left pleura thickened and adherent where he had been wounded. My impression is that the chest wound involved only the thoracic parietes, including the pleura but not the lung.

“At about the end of three weeks he was so far convalescent that we said at our morning visit: ‘You will not need us any more but we will drop in occasionally to see how you regain strength.’ He was able to sit up several times through the day and felt well, had return of appetite, but remarked every day that he could not understand the continuance of the stained sputa. This, from the second day of its appearance, had been not simply rusty, as is usual, but quite red, indeed at times almost jelly-like, and we had frequently discussed the significance of it;

but the most careful daily examination had failed to detect any symptom or abnormal sound, rate or area of dullness not perfectly consistent with the diagnosis of acute unilateral pneumonia. We left him on Sunday morning, he saying 'I will be out in a week at the most, and feel well enough to go now.'

"Upon returning from professional calls, I found an urgent message to go to him but found him dead." The following is Dr. Young's statement:

"While passing through Kinney street near Dr. Woodhull's residence, I suddenly heard my name called loudly, and, turning around saw the call came from Dr. Woodhull's house. Rapidly entering I saw the patient within ten minutes, probably less, after the first seizure, which was described as follows: The patient had been sitting up by the window, when he suddenly complained of feeling faint, without any pain, and was assisted by his wife to bed, when he became immediately unconscious, with slow difficult respiration, and rapidly cyanosed. When seen by me

he was in this condition with hardly any pulse, deeply cyanosed, irregular and slow respiration, eyes rolled upward out of their natural axis, insensible to light; no sign of any convulsive movement. Recognizing the extreme condition of the patient, a free hypodermic injection of ammonia was given with a view, if possible, to stimulate the heart and of its alkalizing effect upon the blood. Within a very short space of time, the cyanosis began to fade and disappear from the forehead down, so that the face assumed a natural hue; the respiratory rhythm was re-established, the pulse came up, so that it became at least distinctly preceptible.

"This condition of apparent reaction, without, however, any sign of returning consciousness (at least as far as muscular manifestations or impressions were concerned) continued for a short time, almost giving hope of a chance for life, but soon he relapsed into the condition, in which he was first found and died."

No autopsy was made. Indeed the absence of records of post-mortem examina-

tions in these cases of sudden death in acute pneumonia forms one of the difficulties in arriving at positive conclusions as to the cause or causes. The clinical history of these cases leave no doubt as to the diagnosis. A fatal termination is to be expected in a certain percentage of cases of acute lobar pneumonia under any circumstances, a larger proportion occurs in private practice, where the surroundings and other considerations of sentiment or convenience forbid the urging of autopsies as matters of simple scientific interest, and thus in most cases we are satisfied with crediting the sudden fatal termination to any of the various lesional possibilities that may occur in pneumonia by embracing them all under the term, heart failure. But, does this explanation explain? Heart-failure as a true direct cause of death can happen in two ways; either by sudden paralysis of innervation through the branches of the pneumogastric nerve supplying it or the inhibitory-ganglia regulating it; or by structural degenerative changes in the heart muscle

itself; in the one case the motor power being lost, in the other the motor machine refusing to act, being worn out. Discarding, hence, the term heart-failure as a pathological entity in explaining sudden death in pneumonia, we are forced to look elsewhere. As a guide in which direction to search for the cause or causes two facts should be borne in mind:

1. That these cases of sudden death under consideration, generally occur during or after apparently fully established convalescence, and

2. That the fatal attack is ushered in suddenly and terminates fatally under tumultuous signs of a struggle between the heart and lungs, indicating plainly the sudden occurrence of mechanical obstructions in the right heart and pulmonary circulation, as shown by the weak and imperceptible pulse, the dyspnoea, irregularity of respiration, the intense and rapid cyanosis, and unconsciousness.

Of all the authors of treatises on pneumonia in the various text-books, so-called, accessible to me the one who comes nearest

to a full appreciation of the true cause of this class of sudden death is Flint. He says: "There is a liability in the course of this disease to an occurrence which claims special notice, namely, coagulation of fibrin in the right auricle or ventricle; that is to heart thrombus; this is of not infrequent occurrence in fatal cases of pneumonia. It occurs especially when an entire lung becomes involved, and in cases of double pneumonitis.

"Thrombi formed ante-mortem and not infrequently the immediate cause of death, are to be distinguished from those produced in the last moments of life and from post-mortem clots. Their formation sometimes may be determined and with much confidence during life. In a case presenting no symptoms which denote imminent danger a sudden change takes place for the worse; the circulation is notably disturbed as shown by the frequency, feebleness and irregularity of the pulse; there is a sense of the want of air; the expression is haggard and anxious, cyanosis is more or less marked; the patient falls

speedily into a moribund state; and this unexpected change is not connected with an extension of the disease to a new lobe or any newly-developed inflammatory complication."

Assuming then that Flint is correct in ascribing heart thrombus as a not infrequent cause of death in pneumonia, although no special emphasis is put on "sudden death" during apparent convalescence, let us extend our inquiry a little further by including embolism of the pulmonary vessels with heart thrombus, or vice versa. If they are found to be the cause of sudden death in numerous and varied other pathological processes there is, for very good reasons to be alluded to further on, no ground to doubt that we here have the principal cause of sudden death in pneumonia. It is not intended to go into an elaborate or exhaustive examination of details. A few facts obtained by a cursory examination of accessible records will illustrate the subject.

Dr. Raywood Johnson in the transactions of the Path. Soc., London, 1889—

(*Schmidt's Jahrb.*, Vol. 229, p. 249,) describes the case of a woman fifty-four years old, with varices of leg and phlebitis, who died suddenly in an attack of dyspnoea, of embolism in the pulmonary artery, six to seven inches in length, resting on the bifurcation.

A case of sudden death in a case of typhoid fever by embolism of the pulmonary artery is reported from the *Prager Vierteljahrsn.*, in *Schmidt's Jahrb.*, Vol. 228, p. 215. Autopsy showed tolerably coherent coagula in the main trunk of the pulmonary artery.

A case of sudden death in consequence of formation of thrombus in the pulmonary artery, occurring in a case of acute exudative pleuritis is reported by Dr. Dujardin Beaumet in *Gaz. de Paris* (*Schm. Jahrb.*, Vol. 171, p. 150) death occurred when patient got out of bed to go to stool.

In a paper on sudden death by pulmonary embolism in cases of inflammatory varicose veins, Dr. Marc Chabenat, of Paris, (1874) collected seven (7) cases. In five (5) cases the autopsy showed presence

of emboli in the right heart and trunk of pulmonary artery; in the other two cases no autopsy was made, but the history of the case was clear as pointing to the same causes. The author calls attention to the fact that the neighborhood of large venous trunks in which the blood flows yet uninterruptedly favors the detachment of an embolism. In all these cases the emboli were large, as large as a finger, and the detachment of the emboli always took place in the convalescent stage on occasion of sudden movements in bed or when getting out of it.

Two cases of sudden death from embolism and thrombus in the pulmonary artery are reported (*Bullet. de la Soc. Anatom., in Schm. Jahrb.*) due in one case (Dr. Cossy's) to inflammation of the crural vein after operation for strangulated hernia; in the second case, reported by Dr. Hegar, due to thrombosis of the left femoral vein after enucleation of a uterine myoma.

Spontaneous extensive thrombosis of the pulmonary artery caused sudden death in a young chlorotic girl, after a short attack

of dyspnœa. Reported by D. H. Rendt, (*Gaz. Hebdom.*, XXXIV, 16, *Schmidt's Jahrb.*, Vol. 215, p. 249). Embolism excluded.

In his inaugural dissertation, Dr. B. L. B. Bang (*Afhandl für Doktorgraden*, *Schmidt's Jahrb.*, 1882, vol. 195, p. 289) a study of twelve cases of embolism or thrombosis of the pulmonary artery concludes that the most dangerous, i. e., the large emboli, mostly occur in the cases in which the clinical histories show no sign of presence of pre-existing cause.

In a paper on "Sudden Death in Pleurisy" by D. E. Weill (*Revue de Med.*, vol. VII, p. 33, *Schmidt's Jahrb.*, vol. 214, p. 30) the author collects twenty-seven cases of sudden death in pleuritis; in eleven of these cases there was thrombus-formation in the right heart and embolism of pulmonary artery or changes in the myocardium; in six, œdema of the lungs; there was one of pericarditis; in two perforation took place into the lung and in seven cases the autopsies showed no certain cause of death. These latter may have been due to direct

heart failure or brain anæmia. In most of these cases death occurred after strong muscular movements—only three died while quietly lying in bed.

Prof. Adolf Lesser of Breslau, in *Vierteljsch fuer faren Mediz., Schm. Jahrb.* vol. 219, p. 74, gives the most important autopsy results in 171 cases of sudden death; only in fifty-three cases an anatomical cause for the suddenness of death was reached; in twenty-one cases there were pressure upon the brain, apoplexy, tumors, hæmorrhage, aneurism; seven were cases of cardiac aneurism; eight times there was suffocative hæmoptysis and bursting of aneurism into trachea; in seventeen cases aneurism was the cause accompanying the fatal result; six were caused by embolism of the pulmonary arteries; in seven cases genuine pneumonia existed, where no direct cause could be found.

In the Medical and Surgical History of the War of the Rebellion, Part 2d, Medical Volume, under the history of acute diarrhœa among the post-mortem appearances noticed, special mention is made of

over thirty-six cases in which "fibrinous heart-clots with or without an admixture of ordinary coagula were observed." Two of these cases are noted as having died "very suddenly" and "suddenly." One of these cases died suddenly of pneumonia, and in looking over the details I find that eleven of these cases of heart clot and clots in the pulmonary arteries credited to acute diarrhœa were shown by the autopsies to have been complicated with pneumonia in various stages of progress and intensity, namely cases 162-330-352-353-360-377-385-412-525-642-749.

A cursory examination of the details of cases of pneumonia given in vol. 3d of the same work, is negative as to the occurrence of ante-mortem thrombosis or embolism and their bearings to sudden death.

If we assume then, that thrombosis or embolism of the heart and pulmonary arteries constitute the true pathology in a certain number of cases of sudden death in pneumonia, as well as in other diseases—an assumption based upon undoubted

facts—we are naturally and logically led to enquire whether the pathological processes and conditions of pneumonia are favorable to their occurrence. Such seems to be the case.

Aside from the undoubted increase of fibrin in the blood in pneumonia, as in acute rheumatism and erysipelas, and its probable greater tendency to coagulation, the pathological processes going on in the minute structure of the lungs in pneumonia themselves are not unfavorable to the occurrence of thrombi and emboli. When the air spaces and bronchi have become fully distended by the exuded inflammatory products and the lung has become solid, when the air vesicles, the air passages, the small bronchi and sometimes the large bronchi are filled and distended with fibrin, pus cells, red blood cells, and epithelium we are told by Delafield and Prudden (Hand-book of Pathology) that “in spite of the pressure on the walls of the air spaces, the blood-vessels in their walls remain pervious.” Hence if even a very minute thrombus shall have acci-

dentally formed in one of the small branches of a pulmonary vein, entering into the formation of the vascular network surrounding the air vesicles, this very fact stated by the eminent pathologists cited, "that the vessels remain pervious" would favor its detachment and chance of entering the current of blood into the right auricle, thence into the right ventricle, there to become entangled in the pectinated muscles or tendinous chords and to form the nucleus of a rapidly enlarging embolic clot extending, as the case may be way into the pulmonary arteries up to their bifurcation, and leading to sudden death.

Another point in the clinical pathology of pneumonia, particularly in infectious pneumonia, which would favor cardiac thrombus and embolus, is the concurrence of endocarditis with the pneumonia. A moderate degree of endocarditis, I think, a much more frequent companion of inflammation of the lungs than is generally supposed and than it is possible to clinically prove in all cases. "So long" we are told

by Delatfield and Prudden "as the endothelial linings of the vessels are intact, a simple retardation of the circulation does not usually suffice to induce coagulation, but changes in the endothelium from a great variety of causes, such as inflammation, degeneration, atheroma calcification and the presence of tumors and foreign bodies favor its occurrence." Thus we see that the existence of endocarditis would favor coagulation.

From what has thus far been stated, it is obvious that whatever means of averting sudden death in pneumonia we may employ, they can only be general and precautionary. The patient should be warned even after the temperature has become normal, not to adopt too soon the habits of fully established convalescence. In fact, he should not be allowed to undergo any muscular exertion or bodily effort, not even the passive one of sitting up, until the expectoration has become of a character to show that the exudative process has entirely ceased and that the degenerative changes in the exudate, neces-

sary for its removal are about completed. The presence or absence of blood, or of its elements in the expectoration may probably be considered an important guide in this direction. Remedial agents should be employed to allay the severity of the cough with its danger that by mere mechanical concussion of the solidified lung any little thrombi in the pulmonary veins may become detached and enter the current of blood toward the heart and resulting in cardiac embolism and emboli of the pulmonary arteries. Ammonia in one form or another should be given during the whole progress of the disease, not only as one of the most valuable heart stimulants that we possess but also to counteract the tendency to coagulation and the hyperinosis, or increase of fibrin in the blood of pneumonia patients. For the suggestion of the probable good effect of ammonia to counteract the tendency to hyperinosis in pneumonia, I am indebted to my friend Dr. Charles Young. As to the particular preparation of ammonia to be used there may be little choice; but

during the last few years my own predilection has been in favor of ammonium salicylate, as it combines the stimulating effect upon the heart with the action of a general blood antiseptic, in cases of infectious pneumonia, and also on account of its good effect in rheumatic endocarditic complication. It can be simply given in ten grains doses every two to four hours.

Having thus opened the discussion on this interesting subject, while I beg that you will pardon me for not having laid before you a more elaborate study, I hope also that you will agree with me, when I say that it should be our duty in every case of sudden death in pneumonia to insist upon a thorough and minute autopsy so that we may know the cause rather than to guess at it.

